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## CLINICAL PROBLEMS OF SCUBA DIVING

by

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## SUMMARY PAGE

### THE PROBLEM

To present a summary of the hazards of SCUBA diving for use of medical practitioners not familiar with the accidents which occur in connection with this type of diving. With the tremendous increase in the number of civilian sports divers, physicians throughout the world will be expected to understand and treat the pathologic results of the pressure exposure encountered in such diving.

### FINDINGS

The major hazards of air embolism and decompression sickness are treated, as well as the minor hazards, such as ear and sinus squeeze or other forms of barotrauma. Methods of prevention and treatment are outlined.

### APPLICATIONS

The information presented in this report will be useful to physicians who may be called upon to treat divers suffering from the effects of unequalized pressure differences across the air-containing structures—middle ears, sinuses, lungs, and gastrointestinal tract.

### ADMINISTRATIVE INFORMATION

This report is submitted as Report No. 8 on Subtask (2) of BuMed Research Project MR005.14-3100 (Physiology of Diving). It is reprint of material presented at a meeting of the Heart Association at Yale University School of Medicine, 21 September 1962, and subsequently printed in Connecticut Medicine (June 1963 issue, Vol. 27, No. 6, Page 312).

# Clinical Problems Of SCUBA Diving

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## *Clinical Problems Of SCUBA Diving*

SINCE the introduction of self-contained underwater breathing apparatus (SCUBA) in the early 1950's, production and use of this diving equipment has increased at a truly fantastic rate. Although precise figures are not available, it is estimated that the number of civilian sports divers employing SCUBA gear number nearly one quarter million people in America today. Considering the physiological range covered by these sports divers, and in view of the inherent dangers of compressed-air diving, the emergence of clinical problems peculiar to SCUBA use was predictable. Because the pathology of hyperbaric exposure is so remote from commonly encountered clinical syndromes, medical practitioners are generally not prepared to cope with the accidents which result from this type of diving. In this context, a brief summary of major clinical problems of SCUBA diving may be in order.

## *Physical Requirements*

Any approach to the medical hazards of compressed-air diving must commence with the basic, preventive question of candidate selection. Unlike the diving medical officer in the Navy, the civilian practitioner is taxed with an extremely difficult range of human beings who present themselves for medical approval relative to diving activities. At first thought, it might seem easy to eliminate all applicants who are overweight, over age, hypertensive, neurotic, or victims of chronic respiratory disorders. In civilian practice, however, the problem is not thus easily resolved. The would-be SCUBA diver will probably pursue his underwater activity, regardless of physical defects determined by examination. The physician, therefore, must accept the role of adviser, shorn of normal dictatorial powers. Enlightened advice, however, may avert casualties,

and such advice is best received from the family doctor.

Evaluation of the potential SCUBA diver should probably be predicated on the likelihood that most candidates will dive regardless, and can best be served by a careful evaluation of the physical disabilities which may limit or preclude this activity. Since most diving casualties relate to abnormalities of the respiratory system, this is a prime object of surveillance. Problems relative to the middle ear and paranasal sinuses can rarely be predicted by ordinary examination. Ability of the individual to equalize pressure in these areas is probably best determined by a test of pressure, in an available chamber, or in underwater trial. In case of the lungs, however, the practitioner has better selective control. Any pulmonary lesion, such as cystic lung disease or healed extensive pulmonary histoplasmosis should be immediately and permanently disqualifying. The reason for this should be made clear to the patient: any condition which could lead to general (as in asthma) or localized trapping of air in the lungs, can result in a fatal case of arterial air embolism with a relatively minor pressure fluctuation. In addition, but of lesser importance, is the fact that impaired pulmonary function predisposes to increased carbon dioxide retention and development of decompression sickness, or "bends". Concerning the cardiovascular system, it may suffice to point out that underwater swimming with SCUBA gear is a most demanding physical exercise, comparing nicely with 440 and 880 yard competitive runs. If the candidate's cardiovascular system is inadequate for such a stress, he should be advised against SCUBA diving. Consideration of other body systems should be predicated on the simple fact that SCUBA swimming is hard work for the entire organism; the candidate should be impressed with this fact. Finally, the doctor and patient alike must

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understand that an underwater environment is both unnatural and hostile, and that any degree of emotional instability will increase the probability of dangerous accident.

### **Major Clinical Hazards**

The major clinical hazards of SCUBA diving are threefold: arterial air embolism, bends, and drowning. Although the terminal fatal event is generally drowning, the proximate cause not infrequently can be traced to air embolism or massive and generalized bends. Because of popular confusion between bends and air embolism, it is important to describe these entities in more detail.

Air embolism occurs when massive quantities of inspired gas are forced through the alveolar cellular wall, to proceed via interstitial pathways toward the mediastinum. In this process, the gas may be forced into the pulmonary venous system, or else dissect into the loose tissues of the mediastinum. Should the gas enter the pulmonary venous system, it will pass directly to the left ventricle, and thence ultimately to the cerebral vessels, with blockage of circulation throughout the circuit. The quantities of gas which enter the circulation under these circumstances are massive, indeed. On at least one occasion, we have calculated more than one liter of air in the heart, aorta, and cerebral vessels of a fatal casualty. Treatment of air embolism consists in *immediate* recompression to a chamber depth of 165 feet, which reduces the intra-arterial gas volume by five-sixths, permitting resumption of normal blood flow. Subsequently over a period of thirty-five hours, reduction of pressure permits safe return to surface environment after the residual gases have been dissolved in the blood stream of the individual, and eliminated through the lungs.

It is unlikely that, in absence of recompression facilities, medical therapy will be of real value in treatment of an actual case of arterial gas embolism. The role of the practitioner, therefore, is that of a preventive adviser, since his judgment of potential pulmonary air trapping may be of critical importance to the diving candidate. In the event of a fatal occurrence of air embolism, however, it is vital that a meticulous autopsy be performed. Presently, SCUBA deaths are invariably labelled as drowning, and autopsies are not obtained; thus, the causal factor of air embolism is not documented. The incidence of fatal embolism cases in the Navy is fortunately small. It follows, therefore, that good information relative to this clinical entity is scarce. Additional autopsy findings which could be acquired in cases of civilian casualties would be of inestimable value in overall evaluation of the syndrome. More importantly, some estimate of the role

of air embolism in SCUBA deaths might be available.

Decompression sickness, caisson disease, or "bends", represents a clinical syndrome quite distinct from arterial air embolism. As previously described, air embolism results from intra-arterial introduction of massive quantities of gas which passes through alveolar walls and terminates as emboli in the cerebral vessels. In the case of "bends", however, a different physical phenomenon is involved. The diver who breathes compressed air under increased ambient pressure will inevitably have a degree of nitrogen dissolved in his blood stream and tissues, depending on the depth and duration of the dive. Upon ascent, this inert gas must be eliminated through the lungs, or else come out of physical solution, producing gas bubbles. Such bubble formation in turn produces intravascular blockage or cellular distortion, either of which will result in tissue anoxia and pain. This pain, commonly centered in areas of poor vascularity, is the characteristic of divers' bends. Most likely sites of election are joint areas of the extremities, with rare involvement of the spinal cord.

In the treatment of "bends," as in the case of air embolism, recompression of the patient is a necessity. Because "bends" does not present a comparable hazard to life, however, a good deal of latitude is allowed in the time interval between accident and pressure treatment. Delays of several hours may be dictated by the distance to nearest recompression chamber. Such a delay will somewhat modify the therapeutic result, but it is important to emphasize that even greatly delayed treatment of bends will generally result in great improvement or total cure. If a delay is anticipated before delivery of the patient to a pressure facility, administration of opiates may be required for control of severe pain, although apparent improvement due to such drugs in no way changes the requirement for recompression treatment.

### **Minor Clinical Hazards**

Although the practicing physician will not often be confronted with major diving accidents, his advice and treatment will frequently be sought in connection with minor casualties of everyday diving. Almost invariably, these derangements will result from unequal pressure differentials in the paranasal sinuses or the middle ear. During descent in the water, external pressure on the body will increase at a rate of about one-half pound for each foot of descent. Clearly, if this pressure is not equalized in the middle ear and sinuses, an unequal pressure differential will exist. In such a case, severe pain will be experienced, and accompanied by extra-

vasation of blood and serum into the closed cavity. Because pressure equalization during ascent is easy and automatic, relief of pain is achieved by return to the surface. The diving enthusiast now presents himself to the physician for diagnosis, treatment, and advice relative to future diving activities. The history of a recent dive, coupled with evidence of extravasated blood in the middle ear or sinuses, will make the diagnosis simple, and in practically all cases, active therapy is not required. The question of future diving activity, however, is not so simply resolved. Generally speaking, occurrence of ear or sinus squeeze at rare intervals should not be contraindication to further diving activities, and might be considered a minor occupational hazard of the sport. Persistent inability to equalize pressure, however, may permanently disqualify the subject for further pressure exposure. In selected individuals, radiation of lymphoid tissues near the eustachian ostia will ultimately permit normal equalization of the middle ear, and suitable therapy for chronic or acute sinusitis may guarantee equalization of these cavities. Generally speaking, the ability to "clear" the ears is a function of time and practice, making prognostication difficult in any event, and occasionally embarrassing.

### Summary

In brief narrative, an attempt has been made to elicit major and minor clinical hazards of SCUBA diving. With the predictable increase in numbers of diving enthusiasts, physicians throughout the

United States will be expected to understand and treat the pathologic results of pressure exposure. Familiarity with these syndromes will improve the effectiveness of the medical practitioner with respect to advice as well as treatment.

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